CHEST Critical Care

Neuroprognostication After Cardiac Arrest

Julie Kromm, MD; Andrea Davenport, MD; and M. Elizabeth Wilcox, MD, PhD

Cardiac arrest is a significant cause of mortality and morbidity. Despite advances in technologies and resuscitative care, patients who remain comatose after cardiac arrest present the bedside clinician with both diagnostic and therapeutic uncertainty because of variable comfort with how best to neuroprognosticate. Recent guidelines attempt to address existing knowledge gaps; however, significant variability remains in clinical practice, including the application of guideline recommendations at the bedside. We present a case-based discussion to illustrate key principles for early care and a subsequent approach to neuroprognostication. We explore many of the clinical nuances in neuroprognostication, including the utility of the clinical examination combined with either neuroimaging or neurophysiologic studies, in helping to care for these patients and support their families in decision-making processes. We discuss how a multimodal approach to neuroprognostication may be subject to site-specific availability of testing. Furthermore, how to incorporate the multidisciplinary team in patient care, including subspecialty services such as neurology and palliative care, is discussed when faced with complex clinical situations.

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KEY WORDS: cardiac arrest; prognosis

Cardiac arrest is common, with an incidence of out-of-hospital cardiac arrest (OHCA) of 47.3 per 100,000 person years and in-hospital cardiac arrest of 1.8 cardiac arrests per 1,000 admissions.[1,2] Approximately 80% of patients remain comatose post-arrest, most commonly as a result of hypoxic-ischemic brain injury (HIBI).[3] Although a small proportion of patients succumb to hemodynamic instability and associated multiorgan dysfunction, the most frequent cause of death post-arrest is the withdrawal of life-sustaining therapies (WLST).[4] As such, neuroprognostication is a crucial component of post-arrest care. When guided by best practice, neuroprognostication is critical in avoiding inappropriate or premature WLST, and its corollary, prolonged and potentially harmful invasive therapies in cases in which a favorable outcome is unlikely.[5]

Clinical Case

A 56-year-old man was found unresponsive surrounded by drug paraphernalia, having

ABBREVIATIONS: CRs = corneal reflexes; FPR = false-positive rate; GCS = Glasgow Coma Scale; HIBI = hypoxic-ischemic brain injury; OHCA = out-of-hospital cardiac arrest; PLRs = pupillary light reflexes; ROSC = return of spontaneous circulation; RPPs = rhythmic and periodic patterns; SSEP = somatosensory evoked potentials; WLST = withdrawal of life-sustaining therapies

AFFILIATIONS: From the Department of Critical Care Medicine (J. K.), Cumming School of Medicine, University of Calgary, Calgary, AB, Canada; the Department of Clinical Neurosciences (J. K.), Cumming School of Medicine, University of Calgary, Calgary, AB, Canada; the Hotchkiss Brain Institute (J. K.), University of Calgary, Calgary, AB, Canada; the Department of Critical Care Medicine (A. D. and M. E. W.), Faculty of Medicine & Dentistry, University of Alberta, Edmonton, AB, Canada; and the Neuroscience and Mental Health Institute (M. E. W.), University of Alberta, Edmonton, AB, Canada.

CORRESPONDENCE TO: M. Elizabeth Wilcox, MD, PhD; email: mwilcox@ualberta.ca

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SHOUT
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IT

suffered an unwitnessed asystolic OHCA. The patient received 10 minutes of CPR and two doses of epinephrine by emergency medical services before return of spontaneous circulation (ROSC). On hospital arrival, the patient's Glasgow Coma Scale (GCS) score was 3 (eye opening, 1; verbal response, 1; motor response, 1), with fixed and dilated pupils.

The underlying cause for his arrest was presumed to be from a drug overdose, because a urine toxicology screen tested positive for opioids, benzodiazepines, and methamphetamines. His ECG, transthoracic echocardiogram, and chest radiograph showed no other contributing pathologic conditions. CT of the head was negative for intracranial pathologic condition. His laboratory test results showed an acute kidney injury and signs of ischemic hepatitis with mild synthetic dysfunction because of his arrest. On arrival in the ICU, his GCS remained 4 (eye opening, 1; verbal response, 1; motor response, 2), and spontaneous jerking movements of his facial muscles and limbs were noted. He was treated with renally dosed levetiracetam, and an EEG was completed urgently that demonstrated generalized slowing and suppression with no epileptiform discharge or seizures.

At 72 h after cardiac arrest, the patient remained comatose with a GCS score of 6 (eye opening, 2; verbal response, 1; motor response, 3). The patient bilaterally presented pupillary light reflexes (PLRs) and corneal reflexes (CRs) and a repeat CT scan of the brain was unchanged from previous. An EEG demonstrated continuous generalized, nonsuppressed slowing. A meeting is planned with the family to determine next steps. What is the prognosis for this patient? How do you discuss your assessment with the patient's substitute decision-maker(s)?

What Are the Goals of Initial Management Post-Cardiac Arrest?

Immediate post-ROSC care focuses on confirming goals of care, stabilization of cardiorespiratory status, and mitigation of secondary brain injury (eg, neuroprotective measures) while concurrently attempting to elucidate the cause of the arrest to identify

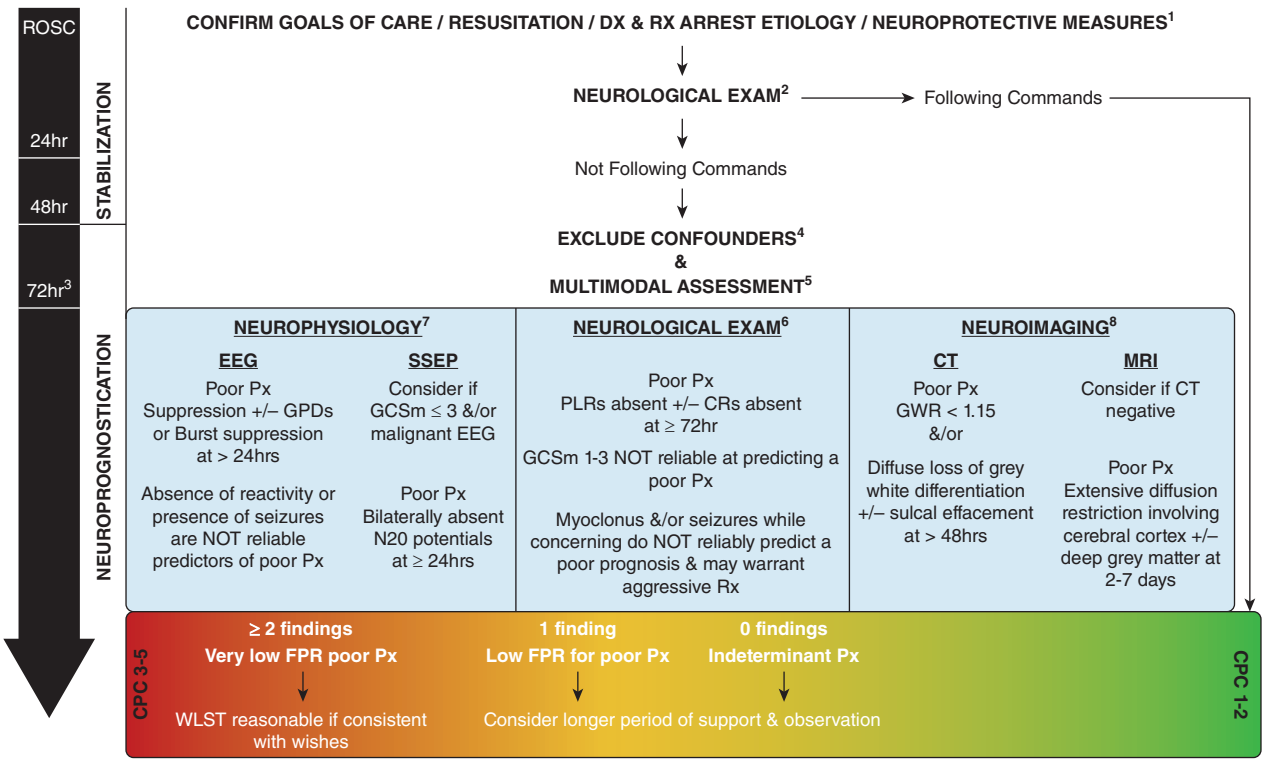
ROSC
24hr
48hr
72hr[3]
STABILIZATION
NEUROPROGNOSTICATION
CONFIRM GOALS OF CARE / RESUSITATION / DX & RX ARREST ETIOLOGY / NEUROPROTECTIVE MEASURES[1]
NEUROLOGICAL EXAM[2]
Following Commands
Not Following Commands
EXCLUDE CONFOUNDERS[4]
&
MULTIMODAL ASSESSMENT[5]
NEUROPHYSIOLOGY[7]
EEG
Poor Px
Suppression +/– GPDs or Burst suppression at > 24hrs
Absence of reactivity or presence of seizures are NOT reliable predictors of poor Px
SSEP
Consider if GCSm ≤ 3 &/or malignant EEG
Poor Px
Bilaterally absent N20 potentials at ≥ 24hrs
NEUROLOGICAL EXAM[6]
Poor Px
PLRs absent +/– CRs absent at ≥ 72hr
GCSm 1-3 NOT reliable at predicting a poor Px
Myoclonus &/or seizures while concerning do NOT reliably predict a poor prognosis & may warrant aggressive Rx
NEUROIMAGING[8]
CT
Poor Px
GWR < 1.15
&/or
Diffuse loss of grey white differentiation +/– sulcal effacement at > 48hrs
MRI
Consider if CT negative
Poor Px
Extensive diffusion restriction involving cerebral cortex +/– deep grey matter at 2-7 days
CPC 3-5
≥ 2 findings
Very low FPR poor Px
WLST reasonable if consistent with wishes
1 finding
Low FPR for poor Px
0 findings
Indeterminant Px
Consider longer period of support & observation
CPC 1-2

Figure 1 – *Multimodal approach to post-arrest neuroprognostication. Details of immediate post-ROSC care. Neurologic examination findings that do not have acceptable false-positive rates for predicting a poor prognosis during early phases of care but indicate which patients warrant further neuroprognostication, timing, exclusion of confounders, multimodal approach, neurologic examination findings, neurophysiologic testing, and neuroimaging are referenced throughout the manuscript. CPC = Cerebral Performance Category; CR = corneal reflex; Dx = diagnosis; FPR = false-positive rate; GCSm = Glasgow Coma Scale motor score; GPD = generalized periodic discharges; GWR = gray-white ratio; PLR = pupillary light reflex; Px = prognosis; ROSC = return of spontaneous circulation; Rx = treatment; SSEP = somatosensory evoked potentials; WLST = withdrawal of life-sustaining therapies.*

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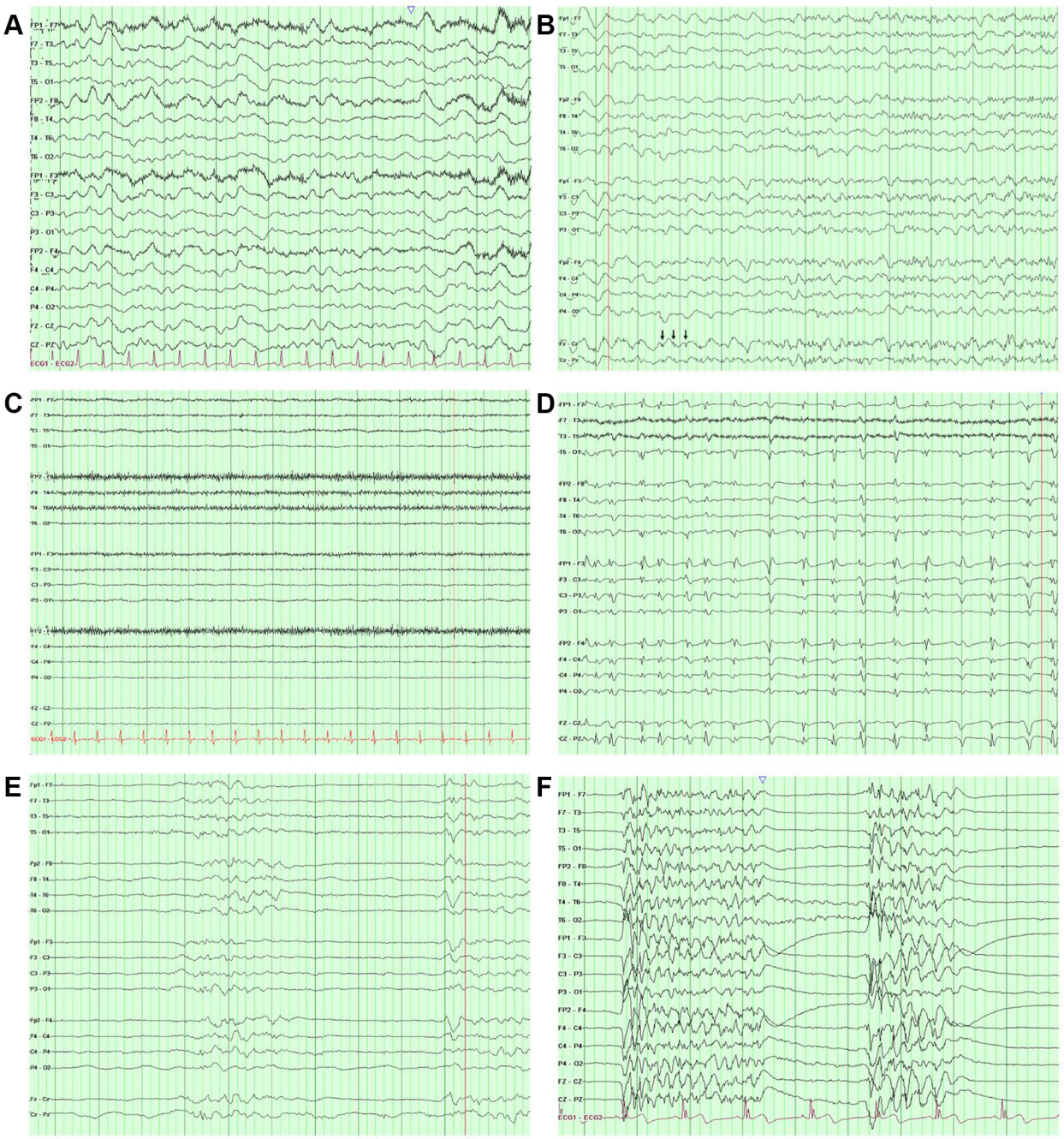

Figure 2 – *EEG demonstrating (A) continuous, generalized, nonsuppressed slowing; (B) generalized slowing with abundant small central and parasagittal epileptiform discharges (arrows), often seen in Lance Adams syndrome, in addition to highly malignant patterns, including (C) generalized suppression, (D) generalized periodic discharges on a suppressed background, (E) nonsynchronous burst suppression, and (F) synchronous burst suppression with identical and highly epileptiform bursts.*

intervenable pathologic conditions (Fig 1).[6] Based on guidelines from the European Resuscitation Council, European Society of Intensive Care Medicine, American Heart Association, and the Neurocritical Care Society, post-cardiac arrest care focuses on resuscitation and subsequent maintenance of a state of normal physiology until further decisions can be made regarding prognosis.[6-8] Neurologic examination findings, lactate levels, and scoring systems that incorporate a variety of prehospital, clinical, and early laboratory values do not have an acceptable false-positive rate (FPR) when predicting a patient's neuroprognosis during this early

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phase of care (Figs 1). Neuroprognostication should be deferred and should not be used for decision-making during this early period or impact decisions to proceed with any invasive procedures as clinically indicated.

When Should Neuroprognostication Be Performed?

Important considerations of neuroprognostication include allowing sufficient time to pass so that confounders can be excluded. After a minimum of 72 h post-ROSC, a comprehensive, multimodal assessment should be completed, including a focused neurologic examination, neuroimaging, or electrophysiologic findings. Guidelines recommend deferral of declarations until at least 72 h post-ROSC in patients who do not undergo therapeutic hypothermia, and 72 h post-rewarming in patients who do undergo therapeutic hypothermia (Fig 1).[8,9] During this time, it is critical to ensure all confounders are minimized or excluded (Fig 1). Sedatives, home medications, and illicit drugs with sedative properties should be afforded sufficient time to clear.[8] It is recommended to wait at least five half-lives to ensure a drug is fully cleared; however, the context-sensitive half-life of sedatives and the influence of renal and hepatic impairment also should be considered.[8] In patients with acute kidney injury, if concerns exist regarding uremia or drug clearance as a potential confounder, a trial of renal replacement therapy should be considered. Testing drug levels, if available, can be helpful to ensure clearance.

When discussing with substitute decision-makers, neurologic outcomes are often dichotomized.[10-12] It is important, however, to remain mindful that scores such as the Cerebral Performance Category scale and Modified Rankin Scale have several limitations, and traditional dichotomies may not align with all aspects of perceived quality of life for each individual patient (Table 1). As such, a discussion of patient values within a context of quality-of-life measures are arguably important in the post-cardiac arrest literature. Exploring patient values at this time is helpful in guiding future discussions to ensure informed decision-making.

Although many prognostic tools have been evaluated in the literature, health care providers should rely on tests and findings with low FPRs and narrow CIs (Fig 1).[8] An FPR of less than 3% to 5% is recognized as clinically acceptable; however, health care providers should use a multimodal approach to identify concordant findings and minimize the potential life-altering risk of a false-positive result. Studies have shown that using a multimodal approach using two or more concordant proven findings, compared with a stepwise algorithm, results in a reduced FPR, near 0 (predicting a patient has a poor prognosis when they do not), and improved sensitivities (ability to conclude that a patient does not have a poor prognosis and thus may have a good prognosis).[13] The best combination of tests to be used in a multimodal approach has yet to be determined and will depend on various factors (accessibility, perceived utility, institutional protocols, patient-specific factors, ongoing confounders, safety, and so forth). Ideally, when using two or more definitive findings to suggest a poor prognosis, they should be from different categories (eg, neuroimaging and physical examination, electrophysiology and neuroimaging), with the caveat that EEG and somatosensory evoked potentials (SSEP) can be considered as separate tests.[8,13]

How to Perform the Neurologic Examination

An unconfounded clinical examination should be performed daily, using proper technique for accurate neuroprognostication (Fig 1).[6-8] The pupillary light reflex, a key component of the physical examination, should be assessed daily. If absent bilaterally at 72 h, it is a reliable predictor of poor functional outcome as part of a multimodal approach to neuroprognostication (FPR, 0-1; 95% CI, 0-8).[10,14] Because of the risk of subjectivity, using quantitative pupillometry as available is recommended.[8] Quantitative pupilometers are small, portable devices that use a combination of infrared and visible light to capture all elements of the PLR, including the maximum and minimum pupil size, constriction percentage and velocity, dilation velocity, and the Neurological Pupil Index (a proprietary algorithm used to provide a numerical understanding of pupillary function on a scale of 0-5). The largest study to date showed that Neurological Pupil Index < 3 at any time between days 1 and 3 had an FPR of 0 with 95% CI of 0-2.[15]

In addition, CRs should be assessed daily. Proper technique is paramount and involves touching the edge of the iris, where the greatest sensitivity is found, then watching for the blink reflex. However, because of high FPRs (4%-10%) with wide CI varying from 0% to 25%, the absence of CRs bilaterally at 72 h post-arrest is unreliable as an independent predictor of poor functional outcome.[8] When both PLRs and CRs are absent bilaterally at $>$ 72 h post-cardiac arrest, this is

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more accurate in predicting a poor prognosis than either finding alone, because the combination of findings helps to mitigate the chance of a false positive from an inaccurate assessment.[6] Other examination findings, including the oculocephalic/vestibulocochlear reflex, gag, and cough reflexes, are not reliable indicators. GCS motor scores of absent (1), extensor posturing (2), or flexor posturing (3) lack specificity, particularly early on, and should not be used to predict a poor prognosis.[8]

Myoclonus is a sudden, spontaneous, involuntary, brief, irregular, shock-like contraction of muscle groups that can be seen post-arrest. It occurs in 16% to 37% of patients and is caused by HIBI-induced hyperactivity of neurons.[8] Although historically viewed as a single entity and an independent indicator of poor prognosis, early-onset myoclonus (diffuse or localized) is no longer recognized in the guidelines as a reliable prognostic tool because significant variability in myoclonus exists, in both its clinical and electrophysiologic presentations.[8,16-20] As an example, status myoclonus is often clinically defined as spontaneous, unrelenting (lasting $>$ 30 min), multifocal, or generalized myoclonus occurring less than 48 hours from ROSC.[8] Lance-Adams syndrome is a form of action- or stimulus-induced myoclonus and is associated with a good neurologic outcome. Clinical features of myoclonus in patients post-cardiac arrest who still have the potential to regain consciousness have been described as asynchronous, multifocal, nonstereotyped (not the same from jerk to jerk), and involving distal limbs. Considering the difficulties that health care providers may have in differentiating status myoclonus from more benign forms such as Lance-Adams syndrome, an urgent EEG should be obtained and consultation with neurology also considered.

Seizures have been reported in 3% to 44% of patients post-cardiac arrest and can be convulsive or nonconvulsive, with only EEG correlates.[21-28] EEG also may reveal rhythmic and periodic patterns (RPPs) that include repetitive uniform discharges in 10% to 35% of patients. These often lie within the ictal-interictal continuum, which is an umbrella term for RPPs that do not meet criteria for electrographic seizures. RPPs are associated with an increased risk for subsequent seizures, secondary brain injury, increased morbidity, and death.[29-33] When detected, uncertainty exists regarding whether treating seizures is beneficial in comatose patients post-arrest.

Patients require an individualized approach in the management of seizures. If seizures or nongeneralized periodic discharge RPPs are identified on EEG after cardiac arrest, particularly when they occur on a continuous, nonsuppressed background, it is reasonable to consider suppressing these patterns with anesthetic or antiseizure medications, with the goal of attenuating secondary brain injury while one seeks out other objective prognosticating indicators (neuroimaging or SSEPs can be particularly helpful because they are not confounded by the sedatives that may be required). If no indicators of a poor prognosis exist, then ongoing aggressive treatment of these EEG patterns should be considered within the context of other medically relevant information (eg, patient age, comorbidities, other active medical issues, and so forth). If other indicators of a poor prognosis exist, possibly the RPP pattern is an epiphenomenon of an injured brain. Consultation from a neurointensivist or neurologist is helpful in management of these complex cases.

How to Incorporate Other Modalities of a Multimodal Assessment

Neurophysiologic Studies

Electroencephalography: Important considerations exist for ordering an EEG that include the influence of confounders and timing of the EEG in relation to the patient's arrest. The most important confounder of an EEG are sedatives (including propofol, benzodiazepines, and ketamine), because at high doses these can produce concerning EEG patterns that may falsely suggest a poor prognosis. Although it is common practice for sedatives to be held immediately before and during an EEG, electrophysiologists do not often consider the context-sensitive half-life of sedative infusions, particularly in the context of renal or hepatic impairment. As such, it is the treating team's responsibility to ensure that when EEGs are ordered for prognostication, they are done when the influence of sedatives is no longer a concern. Although EEGs performed for the purposes of screening for nonconvulsive seizures (often because of clinical myoclonus, seizures, or institutional protocol) are often required less than 24 h post-ROSC, EEGs performed for the purposes of neuroprognostication should ideally be completed beyond 24 to 72 h post-ROSC, because this is the timeframe when most EEG patterns have been shown to correlate with patient outcomes and lower FPRs. Although seizures and status epilepticus are often associated with poor neurologic outcomes, this association is not consistent across all studies.[6-8] EEG reports should be reported in a standardized manner

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using the American Clinical Neurophysiology Society's terminology to facilitate their integration multimodal approach to neuroprognostication.[34] Should questions remain based on EEG reports, bedside providers are encouraged to review the raw EEGs with an electrophysiologist to ensure accurate clinical interpretation (Fig 2).

Somatosensory Evoked Potentials

SSEPs assess the integrity of the dorsal column-lemniscal pathway and determine whether electrical potentials are present at the peripheral, spinal, subcortical, and cortical levels.[35] Most commonly studied is the N20 potential, generated in the primary somatosensory cortex, after an electrical stimulus is applied to the median nerve.[35] Although guidelines recommend various time points (beyond 24 and 48 h), when properly performed and interpreted with no confounders (eg, spinal cord injury), the bilateral absence of N20 potentials when used as part of a multimodal approach is considered a reliable predictor of poor functional outcome (Fig 1).[6-8-35] Other components of SSEPs can be used to prognosticate; however, they have not yet been universally incorporated into guidelines.[35]A detailed review of their use for post-cardiac arrest neuroprognostication has recently been published.[35] Patients in whom SSEPs are most likely to be helpful are those who remain comatose post-arrest, with no purposeful movements to painful stimulation (GCS motor score $\leq$ 3). Several studies have also established that in patients with benign EEG patterns, N20 potentials are universally present and thus SSEPs may be omitted to ensure appropriate resource utilization. In patients with malignant EEG patterns, SSEP testing may add value as a component of multimodal neuroprognostication. Although SSEPs are traditionally considered less confounded by sedatives and opiates compared with EEG or the neurologic examination, these drugs can increase cortical potential latencies and reduce amplitudes. When combined with other important factors that affect SSEP performance and interpretation, this could also hypothetically produce false-positive results and as such treating teams should ensure that these medications are minimized.

Neuroimaging

Imaging for the purposes of neuroprognostication should not occur until at least 48 h after arrest, at which time particular patterns can confer a poor prognosis (Fig 1).[8] CT scan and MRI can be used to evaluate HIBI from a structural perspective. Findings of HIBI on imaging are attributable to cytotoxic edema and require time to evolve, typically days 2 to 7 post-arrest. These findings, if apparent, can be seen reliably on imaging. If neuroimaging was performed within 24 h of the initial presentation to rule out arrest cause, it may need to be repeated. Both CT scan and MRI have low FPR for predicting a poor prognosis; CT scan is more specific and MRI more sensitive for findings consistent with HIBI.[36]

On CT scan, signs of HIBI may include loss of gray-white differentiation in cortical or deep structures, global edema with sulcal effacement, reduced ventricle size, or basal cistern effacement or a pseudo-subarachnoid sign (ie, an apparent increased attenuation within the basal cisterns caused by a decreased attenuation of brain matter attributable to severe cerebral edema simulating true subarachnoid hemorrhage).[36] Studies assessing the accuracy of CT scan in predicting poor outcome have used quantitative determination of the gray-white ratio (ratio of Hounsfield units of gray matter [most often caudate] as compared to white matter [most often internal capsule]) (Fig 3A).[36] Although studies have reported different cut offs (0.91-1.25; mean, 1.15), a low gray-white ratio when used as part of a multimodal approach is predictive of a poor outcome with a low FPR.[36]

If uncertainty remains because of a relatively benign CT scan, given its higher sensitivity, an MRI of the brain should be considered. On diffusion-weighted imaging, MRI apparent diffusion coefficient and fluid-attenuated inversion recovery sequences are best for detecting cytotoxic edema. Diffusion-weighted imaging and fluid-attenuated inversion recovery will demonstrate hyperintensities in affected areas, and apparent diffusion coefficient will demonstrate corresponding hypointensities. Although some studies used quantitative technology to calculate the volume of brain affected, this is often not available. A large burden of HIBI is considered when diffuse bilateral cerebral cortex and deep gray matter (thalami and basal ganglia) are involved. These findings are suggestive of a poor neurologic prognosis (Fig 3B, 3C).[36]

How Do I Contextualize the Results of a Multimodal Assessment?

When two or more concordant findings suggest a poor prognosis, one can be confident with a very low (near 0) FPR of a poor neurologic outcome. If these findings are in keeping with the patient's wishes, transitioning to comfort measures is appropriate. If only one indicator of a poor prognosis is present, the risk of a false-positive error is higher (FPR $<$ 5%), and if no indicators are

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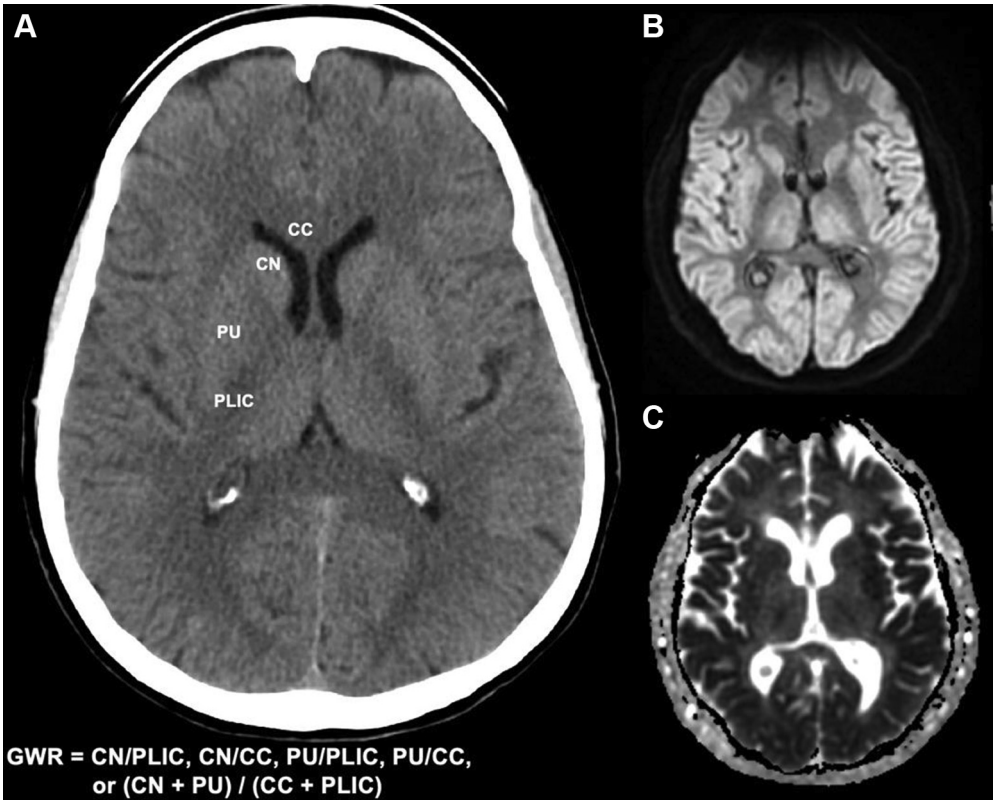

Figure 3 – *Example of a CT head (A) with corpus callosum (CC), caudate nucleus (CN), putamen (PU), and posterior limb of the internal capsule (PLIC) labeled. The gray-white ratio (GWR) can be calculated by measuring the Hounsfield units of the corresponding structures and calculating the ratio according to the equations provided. Example of magnetic resonance brain diffusion weighted imaging (DWI) (B) and corresponding apparent diffusion coefficient (ADC) (C) showing diffuse cortical and basal ganglia DWI diffusion hyperintensities and corresponding ADC hypoin tensities suggestive of a large burden of HIBI.*

present, the patient's prognosis is indeterminant. In the latter two circumstances, a longer period of observation is recommended. The required time for further observation can be variable and would depend on the patient's trajectory. The late awakening prognostic factors and long-term OHCA results of the prospective

TABLE 1] Quantification of Neurologic Function in Cardiac Arrest Survivors

Cerebral Performance Category (CPC)[10]	
CPC 1	Good cerebral performance: conscious, able to work independently, may have mild neurologic deficit
CPC 2	Moderate cerebral disability: conscious, able to work with assistance, able to be independent for activities of daily life
CPC 3	Severe cerebral disability: conscious, dependent on assistance for activities of daily living
CPC 4	Coma or vegetative state, does not meet criteria for brain death, may have spontaneous eye opening and sleep/wake cycles
CPC 5	Death by Neurologic Criteria (DNC)
Modified Rankin Scale[11,12]	
0	No symptoms
1	No significant disability: able to carry out all usual activities and tasks despite symptoms
2	Slight disability: unable to perform all previous activities but able to look after own affairs without assistance
3	Moderate disability: requiring some help but able to walk without assistance
4	Moderately severe disability: unable to walk without assistance and unable to attend to own bodily needs without assistance
5	Severe disability: bedridden, incontinent, and requiring constant nursing care and attention
6	Death

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Norwegian Cardio-Respiratory Arrest Study (NORCAST), a single-center prospective observational study, included patients who remained comatose after ROSC from an OHCA.[37] This study found that 49% and 42% had good outcomes (cerebral performance category 1-2) after median 6 months and 5.1 years, respectively.[37] More than 30% of patients with a GCS score < 9 when sedation was weaned at 72 h post-arrest had a good outcome when given more time to regain consciousness. Although most patients who obtained a good outcome regained consciousness within the first 12 days post-ROSC, a small proportion of patient required up to 25 days post-ROSC.[37]

As previously mentioned, treating teams should not hesitate to engage other experts in the neuroprognostication. Multidisciplinary team involvement including social work, spiritual liaisons, and subspecialty physicians with content expertise is recommended at all stages in the neuroprognostication process. Consultation from either a neurointensivist or neurologist with content expertise also may help navigate challenging clinical cases such as post-arrest myoclonus or status epilepticus and also can provide a second opinion. Palliative care providers also may be able to provide expertise in contextualizing findings within a patient's values and beliefs and explaining next steps.

Clinical Case Conclusion

A meeting with the patient's family is held, and based on the patient's previously expressed wishes and lack of poor prognostic indicators, the decision is made to continue with supportive care. Renal replacement therapy and lactulose for hyperammonemia are initiated. A repeat EEG on day 10 demonstrates ongoing improvement in his background activity and no associated nonconvulsive seizures as a potential confounder. On day 16, the patient regained consciousness and started to follow simple commands. He was subsequently discharged from the ICU and transferred to a neurorehabilitation facility. After 2 months of inpatient rehabilitation, he returned home with few dependencies in his activities of daily living.

Summary

The most common cause of death post-cardiac arrest is WLST based on predicted poor neurologic prognosis. Given its impact on patient outcome, neuroprognostication should be performed by a team of individuals with specific training and expertise to mitigate inappropriate or premature WLST. A multimodal approach to neuroprognostication is preferred, because features of the neurologic examination, neuroimaging, and neurophysiologic assessment when combined afford the treating team a high degree of certainty when the prognosis is poor. When uncertainty exists, further elapse of time to assess potential for neurologic improvement is needed because the different modalities are susceptible to confounders and errors in interpretation as a result. Discussions with surrogate decision-makers should balance the uncertainty of neuroprognostication and patient values when determining goals of care via shared decision-making.

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